Abstract

T cell memory can persist in the absence of antigen. However, some memory cells by default are subject to signals accompanying periodic antigen exposure. OX40 is essential to the extent and persistence of Th2 memory when antigen is re-encountered. In an animal model of allergic asthma, inhibiting OX40/OX40L signaling during the secondary response to inhaled antigen suppressed lung inflammation. Inhibiting OX40 at the time of memory cell reactivation reduced the longevity of memory with further inflammation prevented upon tertiary encounter with antigen.